Is Rhabdomyolysis a Rare Manifestation in Celiac Disease?

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To the Editor: After our first case of celiac disease, presenting with rhabdomyolysis, was published¹ another child has been seen with a similar clinical scenario. She was also a 12-year-old girl that complained of vomiting and diarrhea of one months duration, with weakness of her upper and lower extremities and neck, that started a few days earlier. On physical examination, a failure to thrive was detected (height -4.3 S.D. and weight -2.9 S.D.). A neurological examination revealed muscle weakness in all extremities, with the loss of deep tendon reflexes in the lower extremities. She had iron deficiency anemia, and her serum potassium concentration was very low (1.7 mEq/l). Her serum albumin, aspartat aminotransferase (AST), alanine aminotransferase (ALT) and creatine kinase (CK) levels were 1.6 mg/dl, 78 U/l, 53 U/l and 1858 U/l, respectively. The International normalized ratio (INR) (2.99), responsive to vitamin K administration (1.1), were high. Her ECG findings were suggestive of hypokalemia. Her AST, ALT and CK levels increased, reaching 412 U/l, 270 U/l and 5598 U/l, respectively, on the fourth day following admission. No myoglobinuria was detected, and after determining the positive antigliadin IgA, IgG and antiendomyosial antibodies, a duodenal biopsy was performed, which was interpreted as celiac disease. After intravenous potassium replacement, the patient gradually recovered, and on the sixth day of her hospitalization no neurological abnormalities were detected.

After experiencing our second case of rhabdomyolysis, associated with celiac disease, we were suspicious as to the rarity of this association. It is known that hypertransaminasemia is not a rare occurrence in celiac disease.²³ In two series of adult patients with celiac disease, increased AST and/or ALT levels were reported to be 40.4 and 42%, respectively.²³ In 114 children with celiac disease, hypertransaminasemia was detected in 32% of patients at diagnosis.³ Furthermore, celiac disease may be diagnosed in patients with asymptomatic hypertransaminasemia.⁶ In the hepatocytes, ALT is present in higher concentrations than AST, and therefore, with liver injury, ALT level exceeds that of AST (alcoholic liver disease and cirrhosis are the exceptions). An increase in transaminases, particularly in AST, may reflect, not only the abnormalities in liver, but also in the muscles: rhabdomyolysis is one of the most important causes of hypertransaminasemia.⁷ Speculatively, hypertransaminasemia observed in celiac disease may partly be caused by rhabdomyolysis. The reason there are no reports describing rhabdomyolysis in celiac disease, with the exception of two cases, may probably be due to rhabdomyolysis not always being symptomatic. To the best of our knowledge there have been no reports investigating the CK level, which is specific, even for asymptomatic muscle injury, in celiac disease.

In conclusion, it is emphasized that hypokalemic rhabdomyolysis, in a patient with failure to thrive, should alert the physician for celiac disease. In patients diagnosed with celiac disease, an increase in transaminases may probably be due

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to rhabdomyolysis, rather than to hepatic injury. This is especially possible for patients with hypokalemia and the presence of a high AST to ALT ratio. Rhabdomyolysis can best be diagnosed from the serum CK level. Further studies will be required to detect the role of rhabdomyolysis in hypertransaminasemia in patients with celiac disease.

REFERENCES